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# Barley $\beta$ -glucan reduces plasma glucose and insulin responses compared with resistant starch in men

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#### Abstract

Glucose and insulin responses have been reported to be lowered by acute consumption of soluble oat fiber or high amylose cornstarch. This study sought to determine if barley  $\beta$ -glucan and preformed resistant starch reduced glucose and insulin responses in men independently or if a synergism exists between the two carbohydrate sources. A total of 20 men (10 control, 10 overweight; average body mass index, 23.8 vs 29.0) were fed a controlled diet for 2 days before each treatment containing 75 g available carbohydrate. Fasting subjects consumed 10 treatments consisting of glucose or 1 of 9 muffins containing 3 levels of resistant starch (0.1, 6.1, or 11.6 g/tolerance) and 3 levels of  $\beta$ -glucan (0.1, 3.1, or 5.8 g/tolerance) in a Latin square design. Plasma glucose and insulin responses were determined over 4 hours after each treatment. Compared with controls, overweight subjects had significantly higher mean glucose (5.5 vs 6.0  $\pm$  0.1 mmol/L) (P < .003) and insulin (153 vs 285  $\pm$ 21 mmol/L) (P < .0001) concentrations. Glucose (P < .001) and insulin (P < .003) responses were lower and returned to fasting quicker in the controls than in overweight subjects. The highest  $\beta$ -glucan level was the most effective in lowering glucose (P < .001) and insulin responses (P < .0001). Average glucose (P < .025) and insulin (P < .0001) areas under the curve were lowest after the muffins containing the high  $\beta$ -glucan. Resistant starch content was less effective than  $\beta$ -glucan in reducing glucose or insulin response. Acute consumption of barley  $\beta$ -glucan, but not resistant starch, in muffins was effective in reducing glucose and insulin responses in men who were mildly insulin-resistant.

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Keywords:

Amylose; Insulin; Glycemic response; Insulin resistance; Soluble fiber; Human

# 1. Introduction

Elevated glucose and insulin concentrations are the primary indicators for insulin resistance and type 2 diabetes [1,2]. Insulin resistance is associated with obesity, hypertension, dyslipidemia, glucose intolerance [1,3,4], and type 2 diabetes [1,2]. Abnormal carbohydrate metabolism, especially with respect to elevated glucose or insulin concentrations in the blood, occurs with increasing age

and weight [3,5]. Obesity is associated with decreased ability of the body to control blood glucose with normal levels of insulin [6]. This may also be an early step in the development of non-insulin-dependent diabetes mellitus [6]. Insulin resistance increases as weight increases [3] and is more prevalent in obese subjects (up to 46% in obese subjects compared with 4% in a control population) [4]. It has been estimated that occurrence of insulin resistance increases nearly 20% for each 5% increase in weight over the reported weight at age 20 [3]. Delaying the delivery of glucose through dietary means may assist in the management of insulin resistance [7,8].

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Table 1 Baseline characteristics of control and overweight men (mean  $\pm$  SEM) as selected for the study

	Control (n = 10)	Overweight (n = 10)
Age (y)	$42.2 \pm 2.3^{a}$	$41.5 \pm 2.9^{a}$
Height (cm)	$177.5 \pm 1.8^{a}$	$179.3 \pm 2.8^{a}$
Weight (kg)	$75.3 \pm 1.8^{a}$	$93.1 \pm 3.3^{b}$
$BMI^{b}$	$23.8 \pm 0.4^{a}$	$29.0 \pm 1.0^{b}$
Body fat (%)	$18.0 \pm 1.2^{a}$	$25.2 \pm 2.1^{b}$

<sup>&</sup>lt;sup>a</sup> Means within a row with different superscripts are significantly different (P < .05) based on least-mean squares.

Consumption of foods containing soluble fiber or resistant starch (RS) reduces the risk of chronic disease. Risk factors include reductions in blood glucose and insulin [7-9] and improvement of glycemic control in normoglycemic and diabetic subjects [1,7-10] after consumption of soluble fiber or RS. Glucose and insulin responses (peak and/or area under the curve) have been reported to be lower after a test meal containing soluble fiber, including pectin, Oatrim (oat fiber extract), guar gum, gum tragacanth, and methyl cellulose fibers, when compared with the meal without the soluble fiber [8-12]. Consumption of foods high in amylose or RS decreased postprandial glucose and insulin responses in people with normal as well as those with impaired glucose tolerance [13-15]. The amount of soluble fiber or high amylose starch/RS fed in the acute meal tolerances has varied greatly.

Because both soluble fiber and RS modulate postprandial glucose and insulin response, this study evaluated the postprandial glycemic responses of normal-weight and overweight or obese men after consumption of several levels of RS (from high-amylose cornstarch) and soluble fiber ( $\beta$ -glucan from barley) singularly and combined in the same food product. Barley  $\beta$ -glucan has not been evaluated

for its ability to reduce glycemic parameters, and the potential synergism between RS and barley  $\beta$ -glucan has not been examined in men.

#### 2. Methods and materials

## 2.1. Subjects and study design

The study was approved by the Institutional Review Board of The Johns Hopkins University Bloomberg School of Public Health. Medical supervision was provided by Dr Benjamin Caballero, Division of Human Nutrition, The Johns Hopkins University. A total of 20 men, 25 to 56 years of age, were selected for the study after clinical analysis of fasting blood and urine samples and a medical evaluation of their health history (Table 1). The protocol and purpose of the study were explained to the subjects both orally and in writing. Selection criteria included (1) weight stable for 6 months before the study, (2) normotensive, (3) normal fasting glucose, (4) no history of disease affecting carbohydrate metabolism, (5) taking no medication known to affect glucose or lipid metabolism, and (6) no current disease found by a routine urinalysis and blood screen. Half of the subjects had a body mass index (BMI) lower than 25; the other half had a BMI greater than 27. Control and overweight men were paired for age.

An equilibration diet containing 30% fat, 55% carbohydrate, and 15% protein was fed for 2 days before the day of sample collection to be sure all of the subjects were eating a moderately high carbohydrate diet before the acute meal tests. The menu was designed to exclude foods known to lead to colonic gas production. The menu was identical before each acute test. Body weight was used to determine the energy level given to the subjects, and subjects consumed the same amount of energy during all 10 periods.

Table 2 Carbohydrate composition (g) of the treatments as consumed

	Total carbohydrate <sup>a</sup>	Total fiber <sup>a</sup>	Available carbohydrate	$\beta$ -glucan <sup>b</sup>	$RS^c$
Glucose	75	0	0	0	0
Low $\beta$ -glucan (sp	ent malt barley) <sup>d</sup>				
Low RS	94.6	19.5	75.1	0.67	0
Mid RS	100.6	25.5	75.1	0.72	6.28
High RS	105.9	30.8	75.1	0.66	12.67
Medium β-glucan	n (whole barley flour) <sup>d</sup>				
Low RS	80.3	5.4	74.9	3.12	0
Mid RS	86.1	11.2	74.9	3.47	6.07
High RS	90.6	16.4	74.2	2.84	11.51
High $\beta$ -glucan (w	whole barley flour plus barley $\beta$ -glu	ucan extract) <sup>d</sup>			
Low RS	81.8	6.8	75.0	5.32	0
Mid RS	87.3	12.3	75.0	5.27	5.85
High RS	92.2	17.2	75.0	5.26	11.13

<sup>&</sup>lt;sup>a</sup> Total carbohydrates and fibers were determined by Covance Laboratories Inc.

<sup>&</sup>lt;sup>b</sup> BMI measured as weight/height<sup>2</sup> (kg/m<sup>2</sup>).

b β-Glucan content of the flours and muffins was determined enzymatically using the American Association of Cereal Chemists method 32-23 [16,17].

<sup>&</sup>lt;sup>c</sup> Analysis of the RS added to the muffins was provided by National Starch Co.

<sup>&</sup>lt;sup>d</sup> Ingredient used to prepare muffins in addition to the spent malt barley, whole barley flour, barley  $\beta$ -glucan extract and resistant starch listed previously: wheat flour, baking powder, baking soda, skim milk, corn oil, egg white, and artificial sweetener.

Subjects were weighed before breakfast in the Human Studies Facility 2 days before each acute test. After breakfast, subjects were given prepacked lunch and dinner. They were required to consume all foods and beverages given to them and nothing else unless approved by the principal investigators. Subjects were to record all additional items such as water, noncaloric beverages, salt, and pepper. Blood was collected after a 10-hour fast. Each treatment, glucose solution or test muffins, contained 75 g available carbohydrate (total carbohydrate minus fiber and RS). Three types of muffin varying in  $\beta$ -glucan content were made with either (1) spent malt barley, (2) standard barley flour, or (3) a standard barley flour plus added barley  $\beta$ -glucan extract. Each type of muffin was made with no added RS (Novelose 260, National Starch and Chemical, Bridgewater, NJ) or Novelose calculated to provide 6 or 12 g of RS per 75-g available carbohydrate for a total of 9 muffin preparations. Carbohydrate content of the muffins as eaten is listed in Table 2. All 10 treatments (glucose alone and 9 muffin types) were consumed by all subjects, and the order of consumption was randomized in a Latin square design. The spent malt barley was provided by DeGroen's Micro-brewery, Baltimore, Md. Barley flour was provided by National Barley Foods Council (Spokane, Wash), and the barley extract was provided by Van Drunen Farms (Momence, Ill).

# 2.2. Sample collection and laboratory analyses

Blood samples were collected before treatment and at 0.5, 1, 2, 3, and 4 hours after the treatment was consumed. Glucose was determined on an automated spectrophotometric system (Dade Bering Instruments). Insulin (Diagnostics Products Corporation, Los Angeles, Calif) was determined by radioimmunoassay. Two-hour response areas under the curve (AUCs) were calculated by using the method of Gannon and Nutall [10]. Analyses of the flours' nutrient compositions (total carbohydrate, total and soluble fiber) were determined by Covance Laboratories Inc (Madison,

Wis). The  $\beta$ -glucan content of the flours and muffins was determined enzymatically by AACC method 32-23 [16,17]. Analysis of the RS added to the muffins was provided by National Starch Co.

## 2.3. Data calculations and statistical analyses

Insulin resistance was calculated using the homeostasis model assessment (HOMA = insulin<sup>uU/mL</sup> × glucose<sup>mmol/L</sup>/ 22.5) [18]. In addition, a method using fasting insulin (1) and triacylglycerol concentrations and an index of glucose disposal rates (M) corrected for fat-free mass (ffm) based (Mffm = EXP[2.63-0.28  $\times$  (log insulin<sup>nmol/L</sup>) - 0.31  $\times$  (log triacylglycerol<sup>mmol/L</sup>)]) [19] was also used to determine insulin resistance. Data were analyzed statistically with a mixedmodels procedure for repeated-measures analysis of variance (ANOVA; PCSAS, version 8.0, SAS Institute, Cary, NC). Data were evaluated for the main effects of treatment (glucose or level of RS and  $\beta$ -glucan), group (control vs overweight men), time, and interactions among the main effects. Insulin data were log-transformed before statistical analysis because of nonhomogeneity of variance. Data reported are least-squares means and SEM.

## 3. Results

Plasma glucose responses were significantly different between the groups (control vs overweight; P=.003), but no group-by-treatment interaction (P<.455) was observed. Significant differences were observed at specific times in plasma glucose concentrations after the 10 loads were consumed (time, P<.001; treatment-by-time interaction, P<.001) (Table 3). Overweight men maintained plasma glucose above fasting concentrations longer than did the control men (group-by-time interaction, P<.001). Overweight subjects had significantly higher mean glucose concentrations compared with control concentrations at 0.5, 1, 2, and 3 hours. Both groups' glucose concentrations

Table 3 Glucose responses (mmol/L) after glucose and 9 muffins containing 3 levels of RS and 3 levels of  $\beta$ -glucan consumed in a Latin square<sup>1</sup>

		-		, -		
Treatment	Fasting	30 min	60 min	120 min	180 min	240 min
Glucose	5.21 ± 0.20	$8.36 \pm 0.20^{a}$	$7.55 \pm 0.20^{a}$	$4.67 \pm 0.20^{c}$	$4.20 \pm 0.20^{d}$	$4.73 \pm 0.20^{ad}$
Low $\beta$ -glucan						
Low RS	$5.29 \pm 0.20$	$7.71 \pm 0.20^{b}$	$6.98 \pm 0.20^{ab}$	$5.29 \pm 0.20^{\mathrm{bd}}$	$4.65 \pm 0.21^{a}$	$4.74 \pm 0.20^{ad}$
Mid RS	$5.16 \pm 0.20$	$7.51 \pm 0.20^{b}$	$6.94 \pm 0.20^{ab}$	$5.19 \pm 0.20^{bd}$	$4.87 \pm 0.20^{abe}$	$4.88 \pm 0.20^{abd}$
High RS	$5.26 \pm 0.20$	$7.31 \pm 0.20^{bc}$	$6.80 \pm 0.20^{b}$	$4.95 \pm 0.20^{bc}$	$4.67 \pm 0.20^{a}$	$4.90 \pm 0.20^{abc}$
Medium $\beta$ -glucan						
Low RS	$5.19 \pm 0.20$	$7.38 \pm 0.20^{\rm bd}$	$7.54 \pm 0.20^{a}$	$5.88 \pm 0.20^{a}$	$4.89 \pm 0.20^{abc}$	$4.78 \pm 0.20^{ad}$
Mid RS	$5.22 \pm 0.21$	$7.31 \pm 0.21^{bd}$	$7.30 \pm 0.21^{ab}$	$5.45 \pm 0.21^{ab}$	$4.83 \pm 0.21^{abce}$	$4.87 \pm 0.21^{abd}$
High RS	$5.13 \pm 0.19$	$7.29 \pm 0.19^{bd}$	$6.80 \pm 0.20^{b}$	$5.43 \pm 0.19^{ab}$	$4.63 \pm 0.19^{a}$	$4.67 \pm 0.19^{d}$
High $\beta$ -glucan						
Low RS	$5.14 \pm 0.20$	$6.85 \pm 0.20^{\circ}$	$6.83 \pm 0.20^{b}$	$5.21 \pm 0.20^{bd}$	$5.11 \pm 0.20^{bc}$	$5.06 \pm 0.20^{bc}$
Mid RS	$5.22 \pm 0.20$	$7.04 \pm 0.20^{d}$	$6.67 \pm 0.20^{b}$	$5.62 \pm 0.20^{ad}$	$5.20 \pm 0.20^{ce}$	$5.07 \pm 0.20^{\circ}$
High RS	$5.24 \pm 0.22$	$7.20 \pm 0.21^{bd}$	$6.81 \pm 0.21^{b}$	$5.37 \pm 0.21^{ab}$	$5.26 \pm 0.21^{c}$	$5.12 \pm 0.21^{c}$
ANOVA within a collection time	P = .712	P < .001	P < .049	P = .002	P < .001	P = .001

<sup>&</sup>lt;sup>1</sup> Mean SEM of 10 normal and 10 overweight men. Overall ANOVA: treatment, P = .179; time, P < .0001; treatment-by-time, P < .0001. Means with different superscripts within a column (a-c) are significantly different (P < .05). Low, medium and high β-glucan averaged 0.1, 3.1, or 5.8 g/tolerance, respectively. Low, mid, and high RS averaged 0.1, 6.1, or 11.6 g/tolerance, respectively.

Table 4 Insulin responses (pmol/L) after glucose and 9 muffins containing 3 levels of RS and 3 levels of  $\beta$ -glucan consumed in a Latin square<sup>1</sup>

Treatment	Fasting	30 min	60 min	120 min	180 min	240 min
Glucose	$80 \pm 8^{\dagger}$	495 ± 32 <sup>a</sup>	506 ± 65 <sup>a</sup>	169 ± 33°	79 ± 19 <sup>b</sup>	53 ± 28°
Low $\beta$ -glucan						
Low RS	$70 \pm 8$	$434 \pm 46^{ab}$	$477 \pm 65^{a}$	$242 \pm 33^{abd}$	$124 \pm 19^{ac}$	$59 \pm 28^{ac}$
Mid RS	$68 \pm 8$	$411 \pm 46^{ab}$	$464 \pm 65^{a}$	$275 \pm 33^{ad}$	$137 \pm 19^{ac}$	$115 \pm 28^{b}$
High RS	$76 \pm 8$	$383 \pm 46^{b}$	$475 \pm 65^{a}$	$295 \pm 33^{ab}$	$130 \pm 19^{ac}$	$78 \pm 28^{ac}$
Medium $\beta$ -glucan						
Low RS	$78 \pm 8$	$333 \pm 46^{bc}$	$455 \pm 65^{a}$	$294 \pm 33^{ab}$	$135 \pm 19^{ac}$	$95 \pm 28^{ab}$
Mid RS	$68 \pm 8$	$344 \pm 46^{bc}$	$462 \pm 65^{a}$	$238 \pm 33^{abd}$	$167 \pm 19^{a}$	$80 \pm 28^{abc}$
High RS	$65 \pm 8$	$424 \pm 46^{ab}$	$422 \pm 65^{a}$	$245 \pm 33^{b}$	$107 \pm 19^{c}$	$75 \pm 28^{ac}$
High $\beta$ -glucan						
Low RS	$59 \pm 8$	$263 \pm 46^{c}$	$360 \pm 65^{b}$	$181 \pm 33^{bd}$	$121 \pm 19^{abc}$	$79 \pm 28^{ac}$
Mid RS	$80 \pm 8$	$255 \pm 46^{c}$	$338 \pm 65^{b}$	$224 \pm 33^{ab}$	$142 \pm 19^{ac}$	$101 \pm 28^{ab}$
High RS	$75 \pm 8$	$289 \pm 46^{c}$	$345 \pm 65^{b}$	$207 \pm 33^{\mathrm{bd}}$	$132 \pm 19^{ac}$	$87 \pm 28^{ab}$
ANOVA within a collection time	P = .794	P < .001	P < .001	P < .020	P = .079	P = .035

<sup>&</sup>lt;sup>1</sup> Mean SEM of 10 normal and 10 overweight men. Overall ANOVA: treatment, P < .001; time, P < .0001; treatment-by-time, P < .0001. †Means with different superscripts within a column (a-c) are significantly different based on log transformed evaluation (P < .05). Low, medium, and high β-glucan averaged 0.1, 3.1, or 5.8 g/tolerance, respectively. Low, mid, and high RS averaged 0.1, 6.1, or 11.6 g/tolerance, respectively.

at 0.5 and 1 hour were significantly higher than at all other collection times. Plasma glucose concentrations at 0.5 hour after the glucose treatment were significantly higher and at 2 and 3 hours were significantly lower than concentrations observed after all muffin treatments. The lowest glucose concentrations at 1 hour after the loads were observed after the high  $\beta$ -glucan or high RS treatments.

Insulin responses were significantly affected by group (P < .001), treatment (P < .001), time (P < .0001), groupby-treatment interaction (P < .04), group-by-time interaction (P < .001), and treatment-by-time interaction (P < .001). Overweight men had higher plasma insulin concentrations and maintained them above fasting longer than did the control men. Overweight subjects had significantly higher mean concentrations compared with control concentrations at 0.5, 1, 2, and 3 hours. Overweight subjects had significantly lower mean insulin following the high  $\beta$ -glucan compared with other treatments, whereas the insulin reduction observed in controls did not reach significance. Plasma insulin concentrations were significantly higher at 0.5 and 1 hour than at other times, the response after glucose resulting in the highest concentrations (Table 4). Insulin concentrations at 0.5 and 1 hour after all the high  $\beta$ -glucan loads were significantly lower than concentrations after the other treatments. Plasma insulin concentrations 2, 3, and 4 hours after the muffin treatments were higher than after the glucose load, but a distinct pattern between the different tolerances was not observed.

Differences in the  $\beta$ -glucan and RS content of the loads resulted in a significant difference in glucose AUC (treatment, P < .004) and insulin AUC (treatment P < .0001) (Fig. 1). Both glucose and insulin AUCs were lowest after the treatments containing the highest  $\beta$ -glucan (high  $\beta$ -glucan/low RS, high  $\beta$ -glucan/mid RS, and high  $\beta$ -glucan/high RS). The insulin AUCs after muffins containing the midrange of  $\beta$ -glucan were lower than after the low  $\beta$ -glucan muffins, but the differences were not significant. The insulin AUCs

of overweight men were higher than that of the control men (P < .0001). However, no tolerance-by-group interaction was observed for either glucose (P < .75) or insulin (P < .35). No differences in AUC were observed with varying RS content.

Overweight men had significantly higher mean triacylgl-cyerol concentrations compared with the control subjects (166.6 vs 74.2 mmol/L, respectively; P < .006). Although there were significant differences in treatment (P < .027), group-by-treatment (P < .001), and group-by-time (P < .001), no pattern due to the  $\beta$ -glucan or RS content of the different tolerances was observed. No differences in free fatty acids were observed by group treatment or time.

Insulin resistance calculations resulted in a significant difference between groups with the MFFM method (overweight,  $7.6 \pm 0.22$ ; control,  $9.1 \pm 0.22$ ; P < .0001) or with HOMA (overweight,  $3.0 \pm 0.28$ ; control,  $1.7 \pm 0.28$ ; P < .002). When the fasting insulin values were evaluated based on fasting insulin above or below 87.5 mmol/L [19], 8 of the overweight subjects and 1 control subject were responsible for almost all of the higher values. The HOMA calculations based on grouped fasting insulin rather than weight or BMI resulted in a distinct separation (P < .0001) in insulin resistance; the lower average fasting insulin (62.4 mmol/L) had a value of 1.6, whereas the higher average insulin (125.4 mmol/L) had a value of 4.2.

## 4. Discussion

Glucose and insulin responses have been reported to be improved (lowered or flattened) after a test meal containing a soluble gum, including pectin, Oatrim, guar gum, gum tragacanth, and methyl cellulose fibers, as compared with the meal without the gum fiber [7-9] or insoluble fibers, such as wheat [20]. The addition of soluble fiber from oats [21-24] or guar gum [13] to the diet of adults with type 2 diabetes was beneficial in lowering insulin requirements and/or significantly lower blood glucose concentrations or

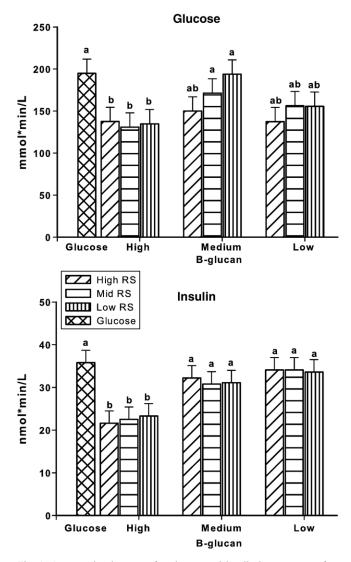


Fig. 1. Areas under the curve for glucose and insulin by treatment after glucose and 9 muffins containing 3 levels of RS and 3 levels of  $\beta$ -glucan consumed in a Latin square. Least-square means  $\pm$  SEM. Area under the curve based on 0- to 120-minute plasma glucose or insulin concentrations. Bars with different superscripts are significantly different (P < .05). Glucose ANOVA: group, P = .13; treatment, P < .004; group-by-treatment, P = .75. Insulin ANOVA: group, P = .0001; treatment, P < .0001; group-by-treatment, P = .35.

postprandial AUCs. In few studies, oat-containing foods have been fed, and glucose and insulin concentrations have not been significantly lowered in normal and hypercholesterolemic subjects [25,26].

Barley has been used in fewer studies as a source of soluble fiber. Lower glucose and insulin responses have been reported after acute consumption of barley pearls, bread, and pasta in normoglycemic [27-29] and type 2 diabetic subjects [30]. The insulin requirement was reduced for some type 2 diabetic subjects when barley was consumed [30]. The amount of soluble fiber consumed in the barley meal affects the postprandial responses. Porridge made with high-fiber barley, but not common barley, significantly lowered glucose and insulin responses compared with white

bread [25]. In a study similar to the one presented here but with women [31], glucose and insulin AUC decreased as the  $\beta$ -glucan content increased; the highest  $\beta$ -glucan content resulted in significant decreases compared with the low- $\beta$ -glucan–low RS muffins.

Tappy et al [21] reported a linear inverse relation between the  $\beta$ -glucan content and the glucose plasma peak and AUC after consumption of 4.0, 6.0, or 8.4 g oat  $\beta$ -glucan. Insulin response did not appear to be dose-dependent. However, Wood et al [32] reported significant reductions in postprandial glucose and also in insulin responses that were inversely linear with the amount (1.8-7.2 g) consumed and with the logarithm of the viscosity of the meals. Delayed or reduced carbohydrate absorption from the gut and not the effects of fermentation was suggested as the mechanism of action of  $\beta$ -glucan in postprandial glucose metabolism [33].

Reductions in postprandial glucose and insulin responses after foods containing 5.8 to 18.4 g of RS have been reported in control, overweight, hyperinsulinemic, and type 2 diabetic subjects [15,34-40]. Behall et al [37] reported a significant reduction of postprandial glucose and insulin concentrations after the consumption of breads containing more than 8 g of RS from high amylose cornstarch. Granfeldt et al [35] reported significantly lower glucose and insulin response curves and AUCs after products containing 12.2 or 18.9 g of RS compared with responses after the standard corn product (2.0 g RS); responses after the 2 high-RS products (different in total and available carbohydrate) were not significantly different. No effect on postprandial glucose, insulin, free fatty acid, or triacylglycerol concentrations occurred after meals containing 0%, 2.7%, 5.4%, or 10.7% of the carbohydrate as RS from high amylose maize [41]. Yamada et al [42] reported significantly smaller postprandial increases in both blood glucose and insulin when subjects with borderline highfasting glucose (111 mg/dL) consumed bread containing 6 g of RS from tapioca. The postprandial responses of the normal group after the 2 breads were not different [42]. None of the subjects reported here had fasting glucose greater than 111 mg/dL during the study. This may have contributed to the lack of response after the different levels of RS in the men.

In a previous study [31], 20 women (10 control, 10 overweight) consumed muffins containing varying amounts of oat  $\beta$ -glucan (assayed to contain 0.7, 3.2, or 8.1 g per average tolerance) and high amylose cornstarch (assayed to contain 0.8, 3.8, or 8.8 g of RS per average tolerance) alone and combined similar to the study reported here. Compared to the muffins containing the lowest amounts of  $\beta$ -glucan and RS, glucose and insulin AUC decreased when  $\beta$ -glucan (17.3%) and 40.9%, respectively) or RS (20.2% and 25.4%, respectively) content increased. Unlike the men reported here, the reduction in glycemic response in the women was enhanced by combining RS and soluble fiber, although they consumed less RS. The greatest AUC reduction occurred after meals containing both high  $\beta$ -glucan and high RS (28% and 49%) lower AUC for glucose and insulin, respectively). Men had the lowest glucose and insulin AUCs after the muffins

containing the high  $\beta$ -glucan regardless of RS content. Controlling the amount of RS in a product is more difficult when high amylose cornstarch or cornmeal is used than with commercially available preparations using preformed RS. The differences between the men and women may have been due in part to the source of the RS. Evaluation of the 2 RS sources in the same subjects would be needed to determine if more preformed RS is needed to match glycemic reduction observed with high amylose corn.

Consumption of 60 g preformed RS (Novelose 260) (rather than high amylose maize cornstarch) for 1 day before an RS/fiber-free tolerance test resulted in significantly lower postprandial plasma glucose and insulin compared with responses after prefeeding the menu without RS [43]. Calculated postprandial insulin sensitivity and C-peptideto-insulin molar ratio was significantly increased following the high-RS diet. No RS effect was observed on plasma triacyglycerol. When a self-selected diet of subjects was supplemented with 30 g of RS (from High-Maize 260) per day, fasting plasma glucose and insulin, as well as glucose AUC after the diets with and without RS, were not different. Insulin AUC was significantly lower, and C-peptide/insulin AUC and total glucose uptake by the adipose tissue were significantly higher after the diet with added RS [44]. No reduction in postprandial glucose or insulin was observed after 30 g of acid denatured crystalline RS mixed with glucose compared with responses after glucose alone [45]. Prefeeding the RS in the diet appears to potentiate a greater postprandial glycemic reduction in a later meal. Improvement of insulin sensitivity occurred after a test breakfast containing at least 6.8 g of RS [36] and chronic RS consumption [43]. Estimates of daily intake of RS range from 3 to 6 g/d in Europe and Australia with similar but inconsistent data for the United States [46].

Similar to soluble fiber, a minimum intake of RS (approximately 5 g or more) appears to be needed, and chronic consumption appears to improve beneficial reductions in postprandial glucose and/or insulin response. Improvement in insulin sensitivity may require more than 7 g of RS or chronic consumption of this type of starch. Current intake estimates of American and European RS consumption are below this level. It appears that more RS than is currently consumed should be included in the diet for these health benefits. Individuals who would benefit the most are those who are overweight, have elevated glucose and insulin, or have reduced insulin sensitivity. Beneficial reductions in glucose and insulin can result when sufficient soluble fiber from isolates or grain sources such as oats or barley is consumed. Consumption of food sources containing adequate levels of  $\beta$ -glucan and RS should reduce the rise of type 2 diabetes.

## References

 DeFronzo RA, Bonadonna RC, Ferrannini E. Pathogenesis of NIDDM. Diabetes Care 1992;15:318-68.

- [2] Haffner M, Stern MP, Mitchell BD, Hazuda HP, Patterson JK. Incidence of type II diabetes in Mexican Americans predicted by fasting insulin and glucose levels, obesity and body fat distribution. Diabetes 1990;39:283-8.
- [3] Bloomgarden ZT. The 1st World Congress on the Insulin Resistance Syndrome. Diabetes Care 2004;27:602-9.
- [4] Vanhala MJ, Pitkajarvi TK, Kumpusalo EA, Takala JK. Obesity type and clustering of insulin resistance-associated cardiovascular risk factors in middle-aged men and women. Int J Obes Relat Metab Disord 1998;22:369-74.
- [5] Kuczmarski RJ, Flegal KM, Campbell SM, Johnson CL. Increasing prevalence of overweight among U.S. adults. The national health and nutrition examination surveys, 1960-1991. JAMA 1994;272: 205-11.
- [6] Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. Committee Report. Diabetes Care 1998;21(Suppl 1):1-29.
- [7] Wood PJ. Cereal-β-glucans: structure, properties and health claims. In: McCleary BV, Prosky L, editors. Advanced dietary fiber technology. Ames (Iowa): Blackwell Science Co; 2001. p. 315-27.
- [8] Wursch P, Pi-Sunyer FX. The role of viscous soluble fiber in the metabolic control of diabetes. Diabetes Care 1997;20:1774-80.
- [9] Behall KM, Hallfrisch J. Effects of grains on glucose and insulin responses. In: Marquart L, Slavin JL, Fulcher RG, editors. Wholegrain foods in health and disease. St. Paul (Minn): American Association Cereal Chemists; 2002. p. 269-82.
- [10] Gannon MC, Nutall F. Factors affecting interpretation of postprandial glucose and insulin areas. Diabetes Care 1984;10:759-63.
- [11] Hallfrisch J, Scholfield DJ, Behall KM. Diets containing soluble oat extracts improve glucose and insulin responses of moderately hypercholesterolemic men and women. Am J Clin Nutr 1995;61: 379-84.
- [12] Hallfrisch J, Scholfield DJ, Behall KM. Physiological responses of men and women to barley and oat extracts (Nu-trimX) II. Comparison of glucose and insulin responses. Cereal Chem 2003;80:80-3.
- [13] Jenkins DJA, Vuksan V, Kendall CWC, Wursch P, Jeffcoat R, Waring S, et al. Physiological effects of resistant starches on fecal bulk, short chain fatty acids, blood lipids and glycemic index. J Am Coll Nutr 1998;17:609-16.
- [14] Behall KM, Scholfield DJ, Yuhaniak I, Canary JJ. Diets containing high amylose vs amylopectin starch: effects on metabolic variables in human subjects. Am J Clin Nutr 1989;49:337-44.
- [15] Behall KM, Howe JC. Effect of long-term consumption of amylose vs amylopectin starch on metabolic parameters in human subjects. Am J Clin Nutr 1995;61:334-40.
- [16] Lee SC, Prosky L, DeVries JW. Determination of total, soluble, and insoluble dietary fiber in foods—enzymatic gravimetric method, MES-TRIS buffer: collaborative study. J AOAC Int 1992;75:395-411.
- [17] Li BW. Analysis of food carbohydrates: sugars, starches, and dietary fiber polysaccharides. In: Tunick MH, Palumbo SA, Fratamico PM, editors. New techniques in the analysis of foods. New York: Plenum Publishing; 1999. p. 193-200.
- [18] Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: insulin resistance and B-cell function from fasting plasma glucose and insulin concentrations in man. Diabetologia 1985;28:412-9.
- [19] McAuley KA, Williams SM, Mann JI, Walker RJ, Lewis-Barned NJ, Temple LA, et al. Diagnosing insulin resistance in the general population. Diabetes Care 2001;24:460-4.
- [20] Leclere CJ, Champ M, Boillot J, Guille G, Lecannu G, Molis C, et al. Role of viscous guar gums in lowering the glycemic response after a solid meal. Am J Clin Nutr 1994;59:914-21.
- [21] Tappy L, Gugolz E, Wursch P. Effects of breakfast cereals containing various amounts of beta glucan fibers on plasma glucose and insulin responses in NIDD subjects. Diabetes Care 1996;19:831-4.
- [22] Anderson JW, Hamilton CC, Horn JL, Spencer DB, Dillon DW, Zeigler JA. Metabolic effects of insoluble oat fiber on lean men with type II diabetes. Cereal Chem 1991;68:291-4.

- [23] Kabir M, Oppert JM, Vidal H, Bruzzo F, Fiquet C, Wursch P, et al. Four-week low-glycemic index breakfast with a modest amount of soluble fibers in type 2 diabetic men. Metabolism 2002;51:819-26.
- [24] Jenkins AL, Jenkins DJA, Zdravkovic U, Wursch P, Vuksan V. Depression of the glucemic index by high levels of β-glucan fiber in two functional foods tested in type 2 diabetes. Eur J Clin Nutr 2002;56:622-8.
- [25] Liljeberg HGM, Granfeldt YE, Bjorck IME. Products based on a high fiber barley genotype, but not on common barley or oats, lower postprandial glucose and insulin responses in healthy humans. J Nutr 1996;126:458-66.
- [26] Cara L, Dubois C, Borel P. Effects of oat bran, rice bran, wheat fiber, and wheat germ on postprandial lipemia in healthy adults. Am J Clin Nutr 1992;55:81-8.
- [27] Wolever TMS, Bolognesi C. Source and amount of carbohydrate affect postprandial glucose and insulin in normal subjects. J Nutr 1996;126:2798-806.
- [28] Yokoyama WH, Hudson CA, Knuckles BE, Chiu MCM, Sayre RN, Turnlund JR, et al. Effect of barley beta-glucan in durum wheat pasta on human glycemic response. Cereal Chem 1997;74:293-6.
- [29] Bourdon I, Yokoyama W, Davis P, Hudson C, Backus R, Richter D, et al. Postprandial lipid, glucose, insulin, and cholecystokinin responses in men fed barley pasta enriched with beta-glucan. Am J Clin Nutr 1999;69:55-63.
- [30] Urooj A, Vinutha SR, Puttaraj S, Leelavathy K, Rao PH. The effect of barley incorporation in bread on its quality and glycemic responses in diabetics. Int J Food Sci Nutr 1998;4:265-70.
- [31] Behall KM, Scholfield DJ, Hallfrisch JG, Liljeberg-Elmstahl H. Consumption of both resistant starch and β-glucan improves postprandial plasma glucose and insulin in women. Diabetes Care 2006;29:976-81.
- [32] Wood PJ, Anderson JW, Braaten JT, Cave NA, Scott FW, Vachon C. Physiological effects of beta-D-glucan rich fraction from oats. Cereal Foods World 1989;34:878-82.
- [33] Wood PJ, Beer MU, Butler G. Evaluation of role of concentration and molecular weight of oat β-glucan in determining effect of viscosity on plasma glucose and insulin following an oral glucose load. Br J Nutr 2000;84:19-23.
- [34] Krezowski PA, Nuttall FQ, Gannon MC, Billington GJ, Parker S. Insulin and glucose responses to various starch-containing foods in type II diabetic subjects. Diabetes Care 1987;10:205-12.

- [35] Granfeldt Y, Drews A, Björck I. Arepas made from high amylose corn flour produce favorably low glucose and insulin response in healthy humans. J Nutr 1995;125:459-65.
- [36] Higgins JA. Resistant starch: metabolic effects and potential health benefits. J OAOC Int 2004;87:761-8.
- [37] Behall KM, Scholfield DJ, Hallfrisch J. Plasma glucose and insulin reduction after breads varying in amylose content. Eur J Clin Nutr 2002;56:913-20.
- [38] Weststrate JA, van Amelsvoort JMM. Effects of the amylose content of breakfast and lunch on postprandial variables in male volunteers. Am J Clin Nutr 1993;58:180-6.
- [39] Noakes M, Clifton PM, Nestel PJ, Leu RL, McIntosh G. Effect of high-amylose starch and oat bran on metabolic variables and bowel function in subjects with hypertriglyceridemia. Am J Clin Nutr 1996;64:944-51.
- [40] Hoebler C, Karinthi A, Chiron H, Champ M, Barry JL. Bioavailability of starch in bread rich in amylose: metabolic responses in healthy subjects and starch structure. Eur J Clin Nutr 1999;53: 360-6.
- [41] Higgins JA, Higbee DA, Donahoo WT, Brown IL, Bell ML, Bessesen DH. Resistant starch consumption promotes lipid oxidation. Nutr Metab 2004;1:8-19.
- [42] Yamada Y, Hosoya S, Nishimura S, Tanaka T, Kajimoto Y, Nishimura A, et al. Effect of bread containing resistant starch on postprandial blood glucose levels in humans. Biosci Biotechnol Biochem 2005;69: 559-66.
- [43] Robertson MD, Currie JM, Morgan LM, Jewell DP, Frayn KN. Prior short-term consumption of resistant starch enhances postprandial insulin sensitivity in healthy subjects. Diabetologia 2003;46: 659-65.
- [44] Robertson MD, Bickerton AS, Dennis AL, Vidal H, Frayn KN. Insulin-sensitizing effects of dietary resistant starch and effects on skeletal muscle and adipose tissue metabolism. Am J Clin Nutr 2005; 82:559-67.
- [45] Ranganathan S, Champ M, Pechard C, Blanchard P, Nguyen M, Colonna P, et al. Comparative study of the acute effects of resistant starch and dietary fibers on metabolic indexes in men. Am J Clin Nutr 1994;59:879-83.
- [46] Goldring JM. Resistant starch: safe intake and legal status. J AOAC Int 2004:87:733-9.